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Kayed et al., J. Mol. Biol., 287(4):781-96, 1999

Chen et al., Progress in Br. Res., 117:327-34, 1998

Lemere et al., Soc. for Neurosci. Abstr., vol 25 pt. 1, Abstr. 519.6, 10/23-10/28, 1999

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Sharon L. Turner, Ph.D.
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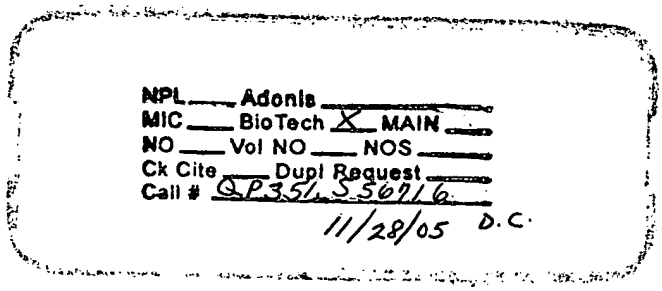

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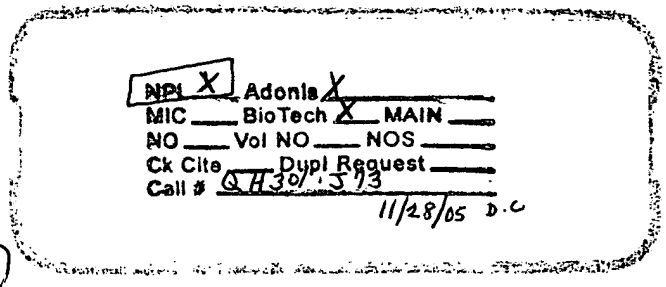

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Kayed et al., J. Mol. Biol., 287(4):781-96, 1999

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ANSWER 4 OF 22 MEDLINE on STN

AN 2004003007 MEDLINE

DN PubMed ID: 14698294

TI Effect of different anti-Abeta antibodies on Abeta fibrillogenesis as assessed by atomic force microscopy.

AU Legleiter Justin; Czilli Dan L; Gitter Bruce; DeMattos Ronald B; Holtzman David M; Kowalewski Tomasz

CS Department of Chemistry, Carnegie Mellon University, 4400 Fifth Avenue, Pittsburgh, PA 15213, USA.

NC AG05681 (NIA)
AG20222 (NIA)

SO Journal of molecular biology, (2004 Jan 23) 335 (4) 997-1006.
Journal code: 2985088R. ISSN: 0022-2836.

CY England: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200402

ED Entered STN: 20040106
Last Updated on STN: 20040211
Entered Medline: 20040210

AB Extensive data suggest that the conversion of the **amyloid**-beta (Abeta) peptide from soluble to insoluble forms is a key factor in the pathogenesis of Alzheimer's disease (AD). In recent years, atomic force microscopy (AFM) has provided useful insights into the physicochemical processes involving Abeta morphology, and it can now be used to explore factors that either inhibit or promote fibrillogenesis. We used ex situ AFM to explore the impact of anti-Abeta antibodies directed against different domains of Abeta on fibril formation. For the AFM studies, two monoclonal antibodies (m3D6 and **m266**.2) were incubated in solution with Abeta(1-42) with a molar ratio of 1:10 (antibody to Abeta) over several days. Fibril formation was analyzed quantitatively by determining the number of fibrils per microm(2) and by aggregate size analysis. m3D6, which is directed against an N-terminal domain of Abeta (amino acid residues 1-5) slowed down fibril formation. However, **m266**.2, which is directed against the central domain of Abeta (amino acid residues 13-28) appeared to completely prevent the formation of fibrils over the course of the experiment. Inhibition of fibril formation by both antibodies was also confirmed by thioflavin-T (ThT) fluorescence experiments carried out with Abeta(1-40) incubated for five days. However, unlike AFM results, ThT did not differentiate between the samples incubated with m3D6 versus **m266**.2. These results indicate that AFM can be not only reliably used to study the effect of different molecules on Abeta aggregation, but that it can provide additional information such as the role of epitope specificity of antibodies as potential inhibitors of fibril formation.

anti-aggregating.

ANSWER 15 OF 22 MEDLINE on STN
AN 2001419655 MEDLINE
DN PubMed ID: 11438712
TI Peripheral anti-A beta antibody alters CNS and plasma A beta clearance and decreases brain A beta burden in a mouse model of Alzheimer's disease.
CM Comment in: Proc Natl Acad Sci U S A. 2001 Jul 31;98(16):8931-2. PubMed ID: 11481462
AU DeMattos R B; Bales K R; Cummins D J; Dodart J C; Paul S M; Holtzman D M
CS The Center for the Study of Nervous System Injury, Washington University School of Medicine, 660 South Euclid Avenue, Box 8111, St. Louis, MO 63110, USA.
SO Proceedings of the National Academy of Sciences of the United States of America, (2001 Jul 17) 98 (15) 8850-5. Electronic Publication: 2001-07-03.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200108
ED Entered STN: 20010903
Last Updated on STN: 20030105
Entered Medline: 20010830
AB Active immunization with the **amyloid** beta (A beta) peptide has been shown to decrease brain A beta deposition in transgenic mouse models of Alzheimer's disease and certain peripherally administered anti-A beta antibodies were shown to mimic this effect. In exploring factors that alter A beta metabolism and clearance, we found that a monoclonal antibody (**m266**) directed against the central domain of A beta was able to bind and completely sequester plasma A beta. Peripheral administration of **m266** to PDAPP transgenic mice, in which A beta is generated specifically within the central nervous system (CNS), results in a rapid 1,000-fold increase in plasma A beta, due, in part, to a change in A beta equilibrium between the CNS and plasma. Although peripheral administration of **m266** to PDAPP mice markedly reduces A beta deposition, **m266** did not bind to A beta deposits in the brain. Thus, **m266** appears to reduce brain A beta burden by altering CNS and plasma A beta clearance.